HUSBANDRY AND HORMONAL FACTORS WHICH INFLUENCE INFLORESCENCE INITIATION IN *Vitis vinifera* cv. CHARDONNAY

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'Let now your breasts be like clusters of the vine...and the roof of your mouth like the best wine.'

Song of Solomon 7, 7-8

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C-16,17 dihydro GA_5, a novel growth retardant was applied to the first 10 developing axillary buds of actively-growing *Vitis vinifera* shoots to investigate the effect on spring shoot growth and inflorescence initiation the following season. Six treatments included 0.00μg, 0.33μg, 1.00μg, 3.33μg, 10.00μg and 33.33μg C-16,17 dihydro GA_5 applied by microdrop directly onto the axillary bud of Mendoza Chardonnay. The following season shoot growth from the labelled buds was measured, as were inflorescence (bunch) number and fruit mass.

The application of C-16,17 dihydro GA_5 had no effect on shoot growth, bunch number or bunch mass the spring following application to developing axillary buds.

Trials were undertaken to investigate the relationship between the developing leaf, and the initiating bud. Defoliation treatments were applied to the first 10 nodes on actively-growing shoots of Mendoza Chardonnay. Treatments included no, alternate and total defoliation. Defoliation consisted of removing the leaf subtending the developing bud by pinching the petiole at the leaf sinus (avoiding damage to the axillary bud). The following season shoot growth from the labelled buds was measured, as were inflorescence (bunch) number and fruit mass.

Total defoliation reduced bud fertility and fruit mass the following season. The rate of spring shoot growth was increased with defoliation. Within the alternately-defoliated treatment these trends were repeated. This suggests the stimulus in grape vines for flower initiation is primarily located in the leaf subtending the axillary bud.

An application of ethephon as 'Ethrel' (500 ppm a.i.) was made to the first 10 node length of actively-growing shoots. The effect on vegetative and fruiting characteristics was to be investigated the following season. Unfortunately the treatment destroyed most of the buds. Those buds that did grow the following spring did so at a slower rate than non-treated buds.

Key words; grape vines, Chardonnay, inflorescence initiation, defoliation, C-16,17 dihydro GA_5, ethephon, axillary bud.

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CHAPTER 1 INTRODUCTION

The aim of this thesis is to:

a) Review the process of inflorescence initiation and bud development in *Vitis vinifera*.

b) Review information of the factors which affect inflorescence initiation in *Vitis vinifera*.

c) Study and discuss the results of experiments examining the role of husbandry and hormonal factors on inflorescence initiation in *Vitis vinifera*. The experiments were conducted over the period of February 1991 to February 1992.

A detailed review of literature is deliberate, ensuring minimal confusion in terminology and time sequences with reference to bud development. It also exposes the lack of information surrounding the precise nature and sequence of mechanisms which activate and regulate inflorescence initiation.

In the experimental section spring shoot growth and bunch numbers were examined after treatments of C-16,17 dihydro GA_5 (a novel GA_3 derived growth retardant), ethephon and manual defoliation of leaves subtending axillary buds.

CHAPTER 2 REVIEW OF THE LITERATURE

2.1 Introduction

Inflorescence initiation is the first step in the reproduction of *Vitis vinifera* (Pratt, 1971) and occurs in the year prior to flowering. Lang (1953) states that in the various stages of flowering floral initiation is by far the most fundamental one for it marks the actual switch from vegetative to reproductive development. The sequence of events which occur during initiation have been described over time (Snyder, 1933; Pratt, 1971) but recent interpretations based on scanning electron microscopy techniques have allowed further detail, demonstrating that the latent bud is a complex 3-dimensional structure and elucidating the origin and morphology of inflorescence (Srinivasan and Mullins, 1981; Scholefield and Ward, 1975). This review of literature endeavours to inspect information on inflorescence initiation of *Vitis vinifera* and study factors which may influence it.

2.2 Morphology of the compound bud

Grapevine buds are generally described as mixed buds in the sense that both leaves and fruit develop from the same bud which forms in a leaf axil (Winkler and Shemsettin, 1937). These buds are also compound buds in consisting of several buds, one located in the axil of another (Srinivasan and Mullins, 1981). The first bud that grows in the axil of a current shoots leaf is called the prompt bud (Srinivasan and Mullins, 1981; Pratt, 1974). It has the potential to grow during the same summer season of formation although it produces inflorescence only occasionally. More often the growth of the prompt bud into the "lateral" or "summer lateral" shoot is inhibited by apical dominance of the summer cane. The first leaf of this lateral shoot is reduced to a bract (sometimes referred to as a prophyll) (Srinivasan and Mullins, 1981; Pratt, 1979) and the meristem in the axil of this bract develops slowly over the summer months into the primary latent bud.

Depending upon the cultivar the latent bud produces 6-10 leaf primordia and up to 3 inflorescence primordia before becoming dormant for the winter (Buttrose, 1969a). Prior to either leaf or inflorescence primordia formation however, the apex of the latent bud produces two or more bracts (similar to the activation in the prompt bud) and in the axils of these form the secondary and tertiary latent buds (Srinivasan and Mullins, 1981). The secondary bud usually bears some inflorescence, although fewer than the primary bud (Pratt, 1974) and the tertiary bud is considered singularly vegetative (Srinivasan and Mullins, 1981). Srinivasan and Mullins (1981) stated that the secondary and tertiary buds rarely exhibit growth due to apical dominance. These three buds, enclosed by the bract of the prompt bud enter dormancy at the end of the growing season and constitute the compound bud or "eye" of the mature wintering cane (Pratt, 1974; Winkler and Shemsettin, 1937). Following initiation during spring and early summer, then winter dormancy the compound bud bursts in the next spring to provide the new season's shoots (Buttrose, 1969a). Thus, the primordia of the inflorescence is formed during the season preceding the year in which the flower blooms (Kliewer, 1987; Pratt, 1979).

2.3 The process of floral initiation/development

Snyder (1933) noted the apical primordium of each latent bud elongates to produce several lateral outgrowths which are the primordia of bud bracts (scales), leaves and inflorescence. By the time approximately four of these have formed, the two alternately located near the base of the bud are elongated and subtly pointed. This is a description reoccurring in literature of primordial leaves. Axillary buds (secondary and tertiary) are laid down in the axils of these modified primordial leaves (also called bracts or prophylls). Additional leaf primordia form simultaneously with elongation of the bud axis. Proximal primordia (near the base of the bud axis) are produced singularly, one per node but distal primordia are produced in pairs. The apical meristem divides into two lobes with each lobe being analogous in the early stage of formation (Snyder, 1933; Barnard, 1932; Pratt

1971; Winkler and Shemsettin, 1937) and often referred to as anlagen - uncommitted primordia (Srinivasan and Mullins, 1981). At the time of origin an anlage is a pad of meristematic tissue (Barnard and Thomas, 1933). Anlagen develop into inflorescence, tendrils or shoots depending on environment and hormonal factors (Srinivasan and Mullins, 1981). Srinivasan and Mullins have demonstrated that anlagen and young tendrils on main and lateral 'summer' shoots have the potential to form inflorescence but this is seldom expressed due to correlative inhibition (which is under the control of hormones) by the respective shoot tips. One lobe develops the pointed tip of a leaf primordia while the other lobe on the opposite side becomes broad and blunt (Winkler & Shemsettin, 1937; Snyder, 1933). This lobe is fated to be either a tendril or a bunch and is always found opposite the foliage leaf (Winkler & Shemsettin, 1937; Buttrose, 1969a; Pratt, 1974). Tendrils are never found below clusters on a shoot and tendril primordia tend to form later in the season than cluster primordia (Buttrose, 1969a;, Winkler and Shemsettin, 1937; Barnard and Thomas, 1933; Antcliff and Webster, 1955).

Barnard and Thomas (1933) stated that differentiation of anlagen into inflorescence primordia takes place during late spring, summer and autumn of shoot growth - while anlage initiated after winter dormancy become tendril primordia. Snyder (1933) when studying Concord (*Vitis labrusca*) found inflorescence initiation to occur in the buds of the summer shoots and continued in the newly forming buds throughout the length of the growing season. However, Lavee *et al.* (1976) only noted differentiation to be "decided" during spring-early summer with development of Sultana (Thompson Seedless) ceasing at about 4 months post bud burst.

The grapevine tendril is universally considered homologous to an inflorescence, but it is only its derivation from morphologically similar anlagen and the development as far as the first bract and arm initiation that is truly similar (Barnard and Thomas, 1933; Pratt, 1974). Other evidence to support the homology of inflorescence and

tendrils are transitional structures, partly tendril-like and partly flower-like (Pratt, 1971; 1974), although usually one or other form predominates (Buttrose, 1969a). Shoots may carry no fruit bunches, in that case tendrils exist where bunches are usually carried (Buttrose, 1969a). Primordia which differentiate as tendrils are usually two or three lobed, the lobes being smooth and the whole structure slender and tapering (Barnard and Thomas, 1933). Barnard and Thomas (1933) described tendrils and the tendril primordium as branch systems in which the branches elongate rapidly but divide only occasionally.

Primordia which differentiate as inflorescence usually develop many lobes, the lobes becoming ridged and the entire structure much wider in relation to its length (Barnard and Thomas, 1933). This obovate primordium is the first visual evidence of a cluster (Snyder, 1933). The second cluster on the lateral bud axis is evident approximately one week after the first. It develops the same way and at the same rate (Snyder, 1933). In buds with more than one inflorescence the proximal one is the biggest (May, 1973; Colby and Tucker, 1926). As it grows, the primordium produces a bract with two arms in its axil (Pratt, 1971; Winkler and Shemsettin, 1937; Snyder, 1933). The outer (abaxial) arm develops into the lowest branch of the inflorescence and the inner (adaxial) arm into the main body of the cluster. The inner arm branches more than the outer arm, with most of the branching occurring over the summer months and slowing down during winter. Barnard and Thomas (1933) describe the growth of inflorescence primordia as a tendency to originate numerous growing apices. A primordium is a complex branch system which resembles a tightly packed bunch of grapes. The primordial branches typically elongate slowly and continue to divide rapidly. Branches ultimately end in flower primordia. Buds cease development and enter organic dormancy over the winter months (Buttrose, 1969a). Barnard & Thomas (1933) noted the small amount of growth which takes place between late summer-autumn and the onset of dormancy does not materially affect the relative development of the anlagen. The growth of the cluster during the dormant period consists of a very slight enlargement of

already existing subclusters (Snyder, 1933). When the buds open in spring however they are in primordial form, with the apical tip of many clusters still an undivided mass of meristematic tissue (Winkler and Shemsettin, 1937; Pratt, 1971). The first visible (by dissection) evidence of flower formation occurs at about the time the buds begin to swell (spring), appearing first and obviously at the base of the cluster. Differentiation of flowers occurs in regular, acropetal order up the cluster immediately following the initiation of flower formation (Snyder, 1933; Barnard and Thomas, 1933). Six to seven weeks after leafing out, the development of flower parts is complete (Winkler and Shemsettin, 1937).

Botti and Sandoval (1990) stated that physiological initiation begins immediately after the first node separates from the shoot apical bud and that inflorescence primordia morphologically (ie. able to be viewed under dissection) appear when there are approximately 10 primordial leaves in the latent bud (cv. Thompson Seedless). Snyder (1933) noted evidence of the first discernable cluster when the shoot was approximately one foot (30 cm) long (cv. Concord). While Pratt (1979) working with Concord noted that within the primary latent bud there seemed to be a correlation between the number of nodes and the first inflorescence primordium. She found the first recognisable inflorescence primordium 0-12 days before bloom, when the shoot had about 13 expanded leaves and 6-7 nodes in the primary bud.

2.4 Variation of inflorescence initiation within vines and between varieties

The origin and morphology of inflorescence in *Vitis vinifera* are similar between varieties. However, variations have been noted in the proportion of buds in which differentiation to flowers occurs and the number of leaf primordia prior to the first inflorescence in the latent bud (Lavee *et al.*, 1976;, Palma and Jackson, 1981). May (1966) states that separate cultivars exhibit different proportions of fruitful buds and even different numbers of primordia per fruitful bud. Changing amounts of radiant energy caused differences between seasons in mean fruitfulness and

trends along cane length. The order and degree of differences changed between varieties (May, 1973).

Differentiation of buds was found to be affected by their positions along the cane (Lavee *et al.*, 1976). A quadratic trend in fruitfulness along the cane was found constant from year to year. The increase from the proximal to the middle position of the cane and then subsequent decrease towards the distal end is thought to be a genetically fixed characteristic (May, 1973). Winkler and Shemsettin (1937) also noted maximal differentiation in the 4th to 12th bud from the head of the cane. From the 12th bud onward the fruitfulness declined. A quadratic trend in size of cluster primordia was noted by Winkler and Shemsettin (1937). The cluster primordia were smaller in the buds at the proximal and distal ends of the cane than in the middle. By the following spring, prior to these buds bursting the magnitude of differences had diminished, yet a small trend persisted.

2.5 Vine vigour and inflorescence initiation

The relationship of inflorescence initiation to vine vigour has drawn varied conclusions. Lavee *et al.* (1976) found no antagonism between vegetative growth and floral initiation in the grapevine. It was shown that both floral induction and differentiation took place at the time of most intensive growth and were correlated with it. They summarised that the development of (inflorescence) primordia is a growth process dependant on vine vigour and not on the process connected with induction and its efficiency. Barnard and Thomas (1933) found the number of anlagen which differentiate to inflorescence were dependant upon the growth rate prior to winter dormancy and the ultimate sizes of the inflorescence were largely controlled by the amount of growth made during the same period. May's (1966) results agree with this, noting that floral initiation follows and may depend on development of leaf primordia in the bud. The bigger buds which were formed on the more vigourous growing shoots gave increased fruitfulness (Buttrose, 1970).

May (1966) could not clearly determine whether increased vine vigour was causal to, or just associated with greater fruitfulness. Lang (1953) noted the time of floral initiation is dependant on the rate of preceding vegetative growth and any condition which may influence this rate may cause a change in the time of flower formation without affecting initiation in any specific manner. Barnard and Thomas (1937) found the fruitfulness of buds in any season was not related to the time of initiation of the first anlage in the developing buds. However, they suggested that conditions which bring about early cessation of shoot growth and rapid accumulation of starch in the canes may be conducive to inflorescence initiation the next season. Statements that carbohydrate accumulation is required for floral initiation (Botti and Sandoval, 1990; Lavee *et al.*, 1976; May, 1965) would support this.

2.6 Factors which influence inflorescence initiation

2.6.1 Metabolic assimilates

Botti and Sandoval (1990) found localised rising starch levels in latent buds would indicate inflorescence initiation. Also an increase in cell, nucleus and nucleolus size of anlagen prior to inflorescence initiation was noted. Lavee *et al.*, (1976) also noted the need for carbohydrate accumulation as a condition for differentiation, and described the relationship between leaves and primordia differentiation as an accumulation of metabolites. May (1965) supports this in stating that a certain level of carbohydrates produced by the leaves under the influence of sunlight, is essential for satisfactory flower production. Photoperiodic stimulus is perceived by the leaf and translocated from there to the apices where flowers are initiated. Thus, light may act on flower induction through the agency of the leaf and the metabolic assimilates it produces (May, 1965).

The abundance of starch grains and the accumulation of labelled carbon in latent buds during inflorescence initiation suggests the accumulation of carbohydrates is integral with successful flower formation in grapevines (Srinivasan and Mullins, 1980a). Sachs (1977) postulates that the effect of chemical or environmental factors on inflorescence initiation may be an indirect result of disturbed metabolic supply and/or distribution to the plant "sinks". Palma (1985) found GA_3 applications gave a greater "efficiency" (measured as dry matter/unit leaf area) of leaves compared to high temperature and auxin (IAA) treatments but less fruitful buds. In contrast to Sachs (1977) he felt the inhibition or promotion of inflorescence on the growing shoot was more the action of plant hormones and not entirely dependant on carbohydrate supply. Perhaps floral initiation may be directed by other factors (possibly hormonal) but certainly the success of inflorescence formation is dependant upon the presence of a minimal amount of assimilates being transported from the leaves to the developing buds. The minute size of an inflorescence anlage in contrast to the entire vine would require very small amounts of carbohydrates to successfully initiate floral production. The carbohydrate localisation is probably a result of other mechanism/s that trigger inflorescence initiation.

2.6.2 Temperature and light intensity

Studies on Sultana (also known as Thompson Seedless) found yields were severely depressed the year after light intensities had been reduced by about 70% for at least 4 weeks prior to flowering (May and Antcliff, 1963). A reduced import of metabolites into the bud when shaded contributed to reduced fruitfulness especially during the period of inflorescence initiation (May, 1965;, May and Antcliff, 1963). May (1965) found that heavy shading of the canopy up to complete darkening consistently reduced the number and size of inflorescence primordia. He noted that unfavourable conditions up to flowering of the current season such as water stress, deficiency of assimilates or hormones, lack of light or low temperatures may all reduce the number of inflorescence formed for the next season. This indicates that

until this stage of growth the developing inflorescence is possibly the weakest "sink" of the growing shoot.

Buds lacking cluster primordia, whether shaded or not, also had smaller leaf primordia than fruitful buds (May, 1965). May suggested that leaf primordia which develop prior to inflorescence primordia 'partly determine' the formation of fruit initials and that shading may reduce bud fertility possibly by affecting the rate of this primordial leaf development. Buttrose (1969a) suggested that the environmental factors promoting primordia differentiation also promote growth in size of the primordia. It is more likely that smaller primordial leaves prior to the floral node is coincidental and not causal to reduced fruitfulness.

It is generally accepted that temperature also influences inflorescence initiation (Buttrose, 1969a, 1969b; Palma and Jackson, 1981). Buttrose (1969b) found the effect of higher temperatures on increased bud fruitfulness was greatest when the node carrying that bud was at the shoot apex. However Palma and Jackson (1981) found a positive correlation of maximum temperature on floral induction when there were three visible nodes above the developing bud on the growing shoot. By the time the bud was positioned 10 nodes back from the growing tip of the cane the effect of temperature upon bud fruitfulness was negligible. Buttrose noted the trend was also similar for light intensity and as the reduction in light intensity resulted in fewer primordia, a reduction in temperature had a still greater effect (Buttrose, 1970). The influence of temperature on the fruitfulness of the buds up to 10 nodes below the shoot apex was proportional to their youthfulness (Buttrose, 1969b). That is, the high temperatures were most effective upon inflorescence initiation at a time when the anlage destined to become the inflorescence did not exist.

Buttrose took this further by calculating approximately three weeks for the separation of 10 nodes on an actively growing shoot. This gave a plastocron of about two days per bud. The time interval for the development of successive buds

was similar to that noted previously (Lavee *et al.*, 1976; Barnard, 1932; Barnard and Thomas, 1933). Thus it appears evident that the buds on a growing shoot differentiate in succession along the length of a growing cane and that any temperature effect occurs well prior to any examinable inflorescence primordia occurring within the developing bud (Buttrose, 1969b).

Buttrose found the total amount of heat energy did not affect inflorescence initiation but evidence suggested a high maximal temperature was conducive to differentiation (Buttrose, 1969b). High temperatures followed by lower temperatures did not exhibit any residual effects of the previous warmer temperature on the differentiating buds. This suggested the warm temperatures acted on the bud *directly* during the early stages of physiological differentiation. May (1965) found complete shading of individual buds to reduce their fruitfulness. May *et al.* (1976) found similarly that individual shoots responded independently and directly to improved light intensity (ie. less shading) and the effect was not due to a "pooled" response from the vine.

Buttrose (1969a) stated that temperature and light intensity can each influence fruitfulness of grapevines although in the field it is possible an interaction exists. He concluded that in the field temperature, rather than light intensity is likely to be of more influence. Both factors are after all a measure of the amount of radiant energy received by a developing bud. Translated into yet another descriptor, Antcliff and Webster (1955) found 'hours of sunshine' in the spring of one season were closely related to the yield in the next season.

2.6.3 Hormones and inflorescence initiation

Gibberellins and Cytokinins

Many naturally-occurring gibberellins exist, with increasing variations of the GA structure continually being added to the nomenclature ($GA_1...GA_n$) as they are discovered and/or synthesised (Crozier, 1983). Only a few gibberellins occur in each plant species. Srinivasan and Mullins (1981) in a review noted the identification of GA_1, GA_3, GA_5 and GA_9 in grapevine xylem sap.

The foremost feature of GAs is their ability to induce vigorous stem elongation and inhibition of flowering in many woody plants (Crozier, 1983;, Jackson and Sweet, 1972;, Weaver and McCune, 1959). This has been found in exogenous applications of GA_3 in grapevines (Lavee *et al.*, 1981). Lavee (1987) found higher levels of endogenous GA in vigorously-growing shoots, especially at the early stages of shoot development during rapid growth. It is suggested gibberellin is required for the initiation of anlagen in *Vitis vinifera* (Srinivasan and Mullins, 1981). The responses of vines to gibberellin (GA_3) and chlormequat (a GA inhibitor) treatments support this (Srinivasan and Mullins, 1980c). The GA-treated anlagen formed tendrils and no inflorescence concluding gibberellins control the formation and growth of floral stems and/or inflorescence axes.

C-16,17 dihydro GA_5 is a naturally occurring plant growth regulator which is synthetically manufactured from GA_3. It was investigated by R. Pharis *et al.* in November 1990 and has been found to inhibit growth in various annual crops (Pharis *et al.*, 1993). A postulated explanation for the cause of growth retardation is that C-16,17 dihydro GA_5 inhibits $[^2H_2]GA_{20}3\beta$-hydroxylation, which consequently yields a low level of $[^2H_2]GA_1$ (Pharis *et al.*, 1993). While the compound successfully retards growth and induces inflorescence initiation in some annual crops, its effect on *Vitis vinifera* is not known. Trials were undertaken to

investigate the effect of C-16,17 dihydro GA_5 on inflorescence initiation and vegetative development on *Vitis vinifera* in the subsequent season.

In the field vines produce many anlagen but only a few develop into inflorescences. Srinivasan and Mullins (1981) suggest endogenous gibberellins occur in ample amounts for anlagen initiation in grapevines. They postulate the limiting factor and principal regulator of anlagen forming inflorescence is the supply of cytokinins (Srinivasan and Mullins, 1980b). Cytokinins have been shown to influence many stages of reproduction in grapevines including inflorescence initiation of the anlagen (Srinivasan and Mullins, 1980b;, Lilov and Andanova, 1976). Conversion of tendrils to inflorescence *in vitro* and *in vivo* with repeated applications of cytokinins further support this. Inflorescence formation is thus a result of weakening apical dominance of the anlagen and tendrils, increasing the branching of these axes and later formation of flowers by cytokinins. Differences between grape varieties exist in the sensitivity of floral response by tendrils to exogenous cytokinin treatments (Srinivasan and Mullins, 1980b). The mobilising effect of cytokinins on latent buds to form inflorescence is so common, Sachs (1977) recommends it as an indication that assimilate supply is an important control of floral development.

Auxins

Auxins, a class of hormones that promote cell elongation also promote the speed of leaf primordia production (shorter plastercron) and increase the number of inflorescence formed in latent buds (Palma, 1975; Palma and Jackson, 1989). Palma and Jackson (1989) suggested that these two responses are under the hormonal control of auxins and are causal to each other. Jindal and Dabas (1982) noted exogenous applications of auxins to Thompson Seedless significantly increased the fruitfulness of buds over the control, but stated only that this was due to changing evidently vegetative buds to floral buds.

2.7 The subtending leaf and its role in inflorescence initiation

The leaf subtending the bud is the receptor of light stimulus (Koblet, 1987). Lavee *et al.* (1976) notes previous work that postulates the biochemical mechanism initiating flower formation is produced in the leaves. They found that in grapevines the stimulus came from leaves located at and above the examined bud. There was no confirmation of the vine "pooling" the stimulus produced in the leaves throughout the vine, thus the presence of leaves is obligatory in the process of floral initiation. Koblet (1987) found leaf removal had no effect on bud fertility the next season, then suggested that initiation had probably already occurred and the leaves were removed too late in the season.

Minnis (1970) working with apricots found removing the leaf subtending a developing bud to inhibit floral initiation. The earlier in the season the leaf was removed the greater was the effect of reduced fruitfulness. It appeared that the factor in the leaf was continuously required throughout the time of inflorescence initiation which led him to postulate that it either was produced in small amounts or was not particularly mobile throughout the plant. Considering the ubiquity and mobility of carbohydrates he believed they only reduced bud fruitfulness when large areas of the tree were shaded. He also dismissed nitrogen compounds as the essential 'floral' factor produced in the leaf. Hormones produced locally in the leaf and in small quantities appealed to Minnis as the most obvious factor for controlling inflorescence initiation.

2.8 Ethrel and ethylene

Ethephon (2-chloroethyl phosphonic acid and also cited as 2-chloroethane phosphonic acid) is an ethylene-releasing compound (Maynard and Swan, 1963; Warner and Leopold, 1969) widely used on crop plants and fruits (Szyjewicz *et al.*, 1984) for a variety of reasons (de Wilde, 1971). Hirshfield and Lavee (1980) found successful ethylene-induced senescence of shoot tip growth in *Vitis vinifera* with "Ethrel", a commercial preparation of ethephon. Ethephon is translocated in plant phloem in a source to sink relationship. Treatment of mature leaves showed ethephon was mobilised to shoot tips (Weaver *et al.*, 1972). The damage to apical meristems and primordia of the growing vine shoot offers an excellent means of vigour control. Ethephon has shown to be more efficient and uniform in vigour control than other registered growth regulators (Hirshfield and Lavee, 1980; Lavee *et al.*, 1977) although time of application may affect the degree of vegetative inhibition, vine yield and fruit composition (Szyjewicz *et al.*, 1984; Weaver and Pool, 1971). Inhibition of vegetative growth has been found to be successful both at late season applications (post-set) and earlier in the season (pre-bloom) (Schulman *et al.*, 1980; Weaver and Pool, 1971). Post-treatment field environment is an important factor as increasing temperature increases the rate of ethylene evolution (Olien and Bukovac, 1978). The success of vegetative inhibition is concentration dependant and related to vine vigour (Szyjewicz *et al.*, 1984; Weaver and Pool, 1971).

With ethephon causing cessation of shoot tip growth studies have reported varied effects on lateral buds. Szyjewicz and Kliewer (1983) and Weaver and Pool (1971) reported the stimulation of lateral buds as metabolites were redirected to mostly summer lateral growth. Other work on grape vines has reported inhibition of lateral buds (Hirshfield and Lavee, 1980; Lavee *et al.*, 1977; Schulman *et al.*, 1980; Wolf *et al.*, 1986) with ethephon treatments. Reduced cold tolerance of the

latent buds has been noted in the winter following Ethrel treatment (Clore and Fay, 1970), although Szyjewicz *et al. (1984)* quotes Pool finding decreased frost damage the winter following high rate treatment. The effect of ethephon on inflorescence initiation varies in many species of plants. De Wilde (1971) reviewed applications of ethephon to apple and pear trees noting retarded vegetative growth and induced flower bud formation. Weaver and Pool (1971) found bud break to be delayed and number of clusters reduced on vines in the season following ethephon treatments. Schulman *et al.* (1980) found no deleterious effects on bud break, growth and cluster numbers the following season, although applications made prior to flowering (anthesis) lowered yield in both current and following seasons. Szyjewicz *et al.* (1984) in reviewing publications of ethephon and the influence in viticulture notes the conflicting results of cold tolerance, the increased viability of dormant buds and delay of bud burst in various cultivars. However there is no mention of the effect of ethephon on inflorescence initiation.

The reduction of auxin synthesis and flow rate by exposure of plants to ethylene is known (Hirshfield and Lavee, 1980; Osborne, 1989). Hirshfield and Lavee (1980) felt that this could lead to the inhibition of vegetative growth found in grapevine treatments. Weaver and Pool (1971) postulated the increase in ethylene/auxin ratio may have been responsible for leaf abscission. If auxin levels are as Palma and Jackson (1989) suggested important in the number of inflorescence initiated in the developing latent buds of grapevines, the effect ethylene has on endogenous auxin levels may affect floral initiation.

2.9 Conclusion

The mechanism which decides whether the anlage opposite the leaf primordia forms a tendril or fruit bunch is largely unknown. May (1965) proposed that it is at least partly determined by the rate of development of the leaf primordia on nodes preceding the 'floral node'. Popular opinion to the cause of floral primordia

formation includes the movement of a floral stimuli (Florigen) from the leaves to the shoot apex (Crozier, 1983). Despite evidence of a floral stimulus, its chemical nature remains unknown and support for the concept of a sole compound responsible for floral initiation has waned (Jackson and Sweet, 1972; Zeevart, 1976). Factors initiating differentiation produced in the leaves seem to behave differently in various species (Lang, 1953). Lavee *et al. (1976)* could not confirm any chemical impulses moving down one branch and up another (cultivars Sultana and Alphonse Lavallee). It appears that the floral induction impulse comes only from leaves located at and above the bud examined (Lavee *et al.*, 1976; Jackson and Sweet, 1972). Any translocation of materials which induce inflorescence initiation from the base of the shoot upwards was not proven. Similarly, Buttrose (1969b) found in temperature treatments, the bud responded directly during an early stage of development and not by initiating the production of a floral stimuli in the plant which then circulates. This strengthens Sachs (1977) view of flower initiation as a result of the modification of metabolic supplies and distribution to the differentiating anlagen by chemical (hormonal) changes influenced by the environment. Sachs (1977) did not believe any single substance acted on the apical meristem of the latent bud to induce flowering. Srinivasan and Mullins (1980c) stated the effects of temperature were mediated through changes in endogenous plant hormones and these directly affected inflorescence initiation.

It is known that the environment, supply of metabolic assimilates and interplay of plant hormones influence inflorescence initiation in *Vitis vinifera*. The exact order and mechanism by which this is done remains ambiguous, if in fact there is a prescribed mechanism.

CHAPTER 3 MATERIALS AND METHODS

Two trials were established with *Vitis vinifera* cultivar Chardonnay at Lincoln University Horticultural Research Area, Canterbury in 1991. The experiments investigated the effects of novel growth retardant C-16,17 dihydro GA_5 on vegetative and floral development. A second trial investigated the effects of ethephon and also various levels of defoliation on vegetative and floral development.

3.1 Experimental site

The vine rows for both experiments contained six-year-old Chardonnay vines (clone Mendoza - sourced from St Helena Vineyard, Canterbury) on Templeton silt loam soil. The vines were trained on a standard upright trellis running north-south with a planting density of 1 x 2 metres. They were annually pruned to two canes and two, two-bud spurs. Each spring the vines were kept to 36 buds per vine with any excess buds rubbed off as they burst.

The site was of moderate vigour with summer drip irrigation used. Active vine growth throughout the length of the season, particularly at the south end of the vineyard row where there was a slight topographical depression was noted. It was assumed that water stress at the northern end of the vine rows accounted for the early cessation of active growth noted in these vines.

3.2 Experimental layout/design

One vineyard row was used per trial. In both experiments actively-growing shoots (where tendrils are longer than the developing shoot apex) were chosen at random within the vineyard row. These were tagged with colour-coded tape at the 10th bud from the tip of the growing shoot. The apical bud was discernable when the leaf

subtending it had separated from the apex. Thus, a tagged cane and its 10 numbered buds followed the pattern illustrated in Figure 3.1 (page 24).

Actively-growing shoots were chosen as buds develop in acropetal order along the length of the cane. The 10 terminal buds of an actively-growing shoot should theoretically exhibit various stages of morphological development at the time of experimental treatment.

For both trials the main plot was arranged in a completely randomised design. Each trial also consisted of a split-plot with treatment of C-16,17 dihydro GA_5, ethephon or defoliation the main plot, and bud position (1-10) on the treated cane was assumed to be the sub-plot. The bud position was not always the sub-plot ie. when the linear effect of bud position was assessed.

3.3 Trial 1 - C-16,17 dihydro GA_5 treatments

Microdrops (10μl) of C-16,17 dihydro GA_5 in a solution of 95% ethanol were applied individually to the 10 buds of a tagged growing shoot. The applications were made on February 8, 1991. The C-16,17 dihydro GA_5 treatments were in logarithmic increments:

1. 0.00μg C-16,17 dihydro GA_5 (95% Ethanol)
2. 0.33μg C-16,17 dihydro GA_5
3. 1.00μg C-16,17 dihydro GA_5
4. 3.33μg C-16,17 dihydro GA_5
5. 10.00μg C-16,17 dihydro GA_5
6. 33.33μg C-16,17 dihydro GA_5

The C-16,17 dihydro GA_5 treatments were applied randomly along the vineyard row. Each treatment had five replicates. A treated cane (refer to Figure 3.1) was in effect a replicate. Insufficient actively-growing shoots per vine allowed one to three treatments per vine. In Section 4.1.2 it is confirmed that treated shoots act independently and are not affected when more than one treatment is made to a vine.

3.4 Trial 2: ethephon and defoliation

The second experiment was applied to Chardonnay vines adjacent to Trial I. The treatments were applied to the first 10 buds of tagged actively-growing shoots as described previously (refer to Section 3.2) and illustrated in Figure 3.1, on February 8, 1991. There were six replicates of each treatment. The treatments were:

1. Control. The 10 buds of the tagged shoot received no treatment (Figure 3.1).

2. Ethephon was sprayed over the tagged cane to drip point. The ethephon was applied at 500mg/l (500ppm) active ingredient using 'Ethrel 48' (48% active ethephon) with a hand held sprayer.

3. Partial Defoliation. The leaves subtending the buds 2, 4, 6, 8, 10 were removed by pinching the petiole at the leaf sinus (avoiding damage to the axillary bud) (Figure 3.2).

4. Total Defoliation. The leaf subtending each labelled bud (ie. buds 1-10) was removed in the same manner as mentioned above (Figure 3.3).

3.5 Data collection

The data collection procedure was identical for both trial 1 and 2.

3.5.1 Freezing microtone: (winter inflorescence count - Trial 1)

In July 1991 dormant buds 3 and 6 were removed from each cane in Trial 1, dissected by freezing microtone (Palma, 1985) and observed under a microscope to obtain inflorescence numbers prior to bud burst. Although inflorescence could be counted in this manner, no differences in numbers between C-16,17 dihydro GA_5 treatments were found. It was found that lesser-developed second and third inflorescence in the compound buds were possibly being destroyed in cross sectioning by the freezing microtone. The remaining buds were left intact on the canes until they grew the following spring when the inflorescences were counted while growing on the vines.

3.5.2 Spring shoot growth

Spring shoot growth was measured at weekly intervals for both experiments. This commenced approximately one week after bud burst and continued for five weeks. The dates of shoot measurements were:

1. 18/10/91
2. 25/10/91
3. 02/11/91
4. 09/11/91
5. 17/11/91

Each treated bud that grew a shoot was measured to the nearest 5mm. Buds that were lost to frost damage over the winter were recorded as missing data throughout the experiment. Buds that grew but were later lost to wind damage were also recorded as missing data. Blind buds (ie. buds that remained intact but did not grow the following spring) were recorded as having a shoot length of '0mm'.

The shoot length was summed for each bud across the length of the cane (ie. buds 1-10) to give total shoot length (mm) at each weekly interval.

3.5.3 Bunch number

On 26 February 1992 all fruit was harvested for both Trials 1 and 2. The number of bunches produced per treated bud was counted. Buds lost to frost and wind damage and also blind buds were recorded as missing data. Buds that grew the following spring and produced only vegetative shoots recorded '0' bunches.

3.5.4 Bunch mass

Bunch mass was measured as grams per shoot (ie. mass of fruit produced by a treated bud that successfully grew the following spring). Buds lost to wind and frost damage and blind buds were recorded as stated in Section 3.5. Buds that produced a shoot but no fruit were recorded as having a fruit mass per growing shoot of 0g.

Average fruit mass (g) per growing shoot was favoured to total fruit mass per treated cane (ie. sum of buds 1-10) as missing shoots (lost to frost, wind and blind buds) reduced the potential harvest of some canes considerably. The mean mass (g) per growing shoot accounted for the mass of fruit harvested from buds that actually grew the following spring.

3.5.5 Bud location on the cane

At the time of experimental applications it was assumed that the proximal treated buds were older than the distal buds. In Trial 2 the effect of bud location on the cane was investigated by comparing buds 1 and 2 (proximal) with buds 9 and 10 (distal). Where damage had occurred (due to frost or wind) and a bud was missing, the adjacent treated bud was measured.

3.6 Statistical analysis

Data for Trials 1 and 2 were analysed using MINITAB and GENSTAT statistical packages.

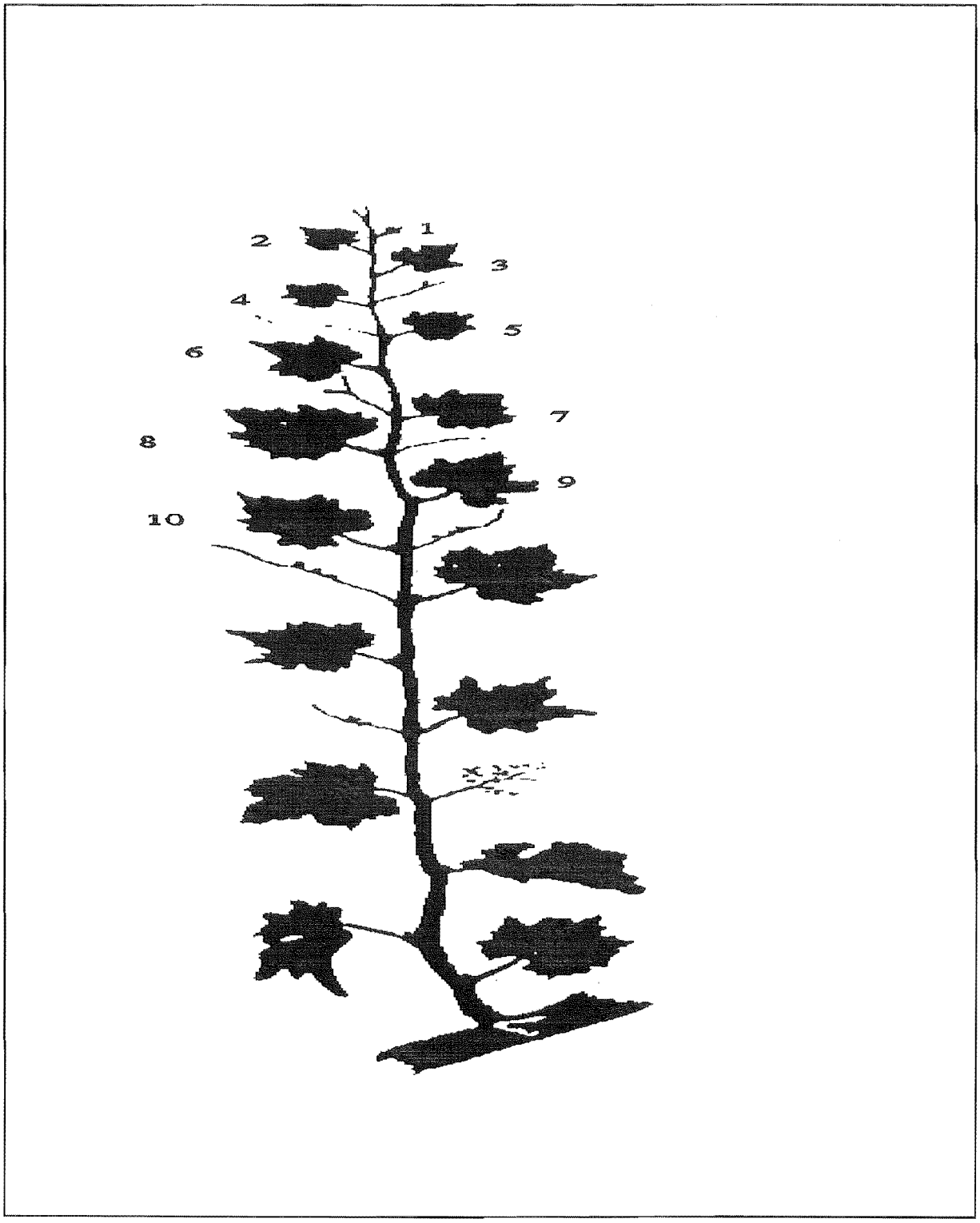


Figure 3.1
Axillary bud numbering system of actively-growing shoots for trials 1 and 2.

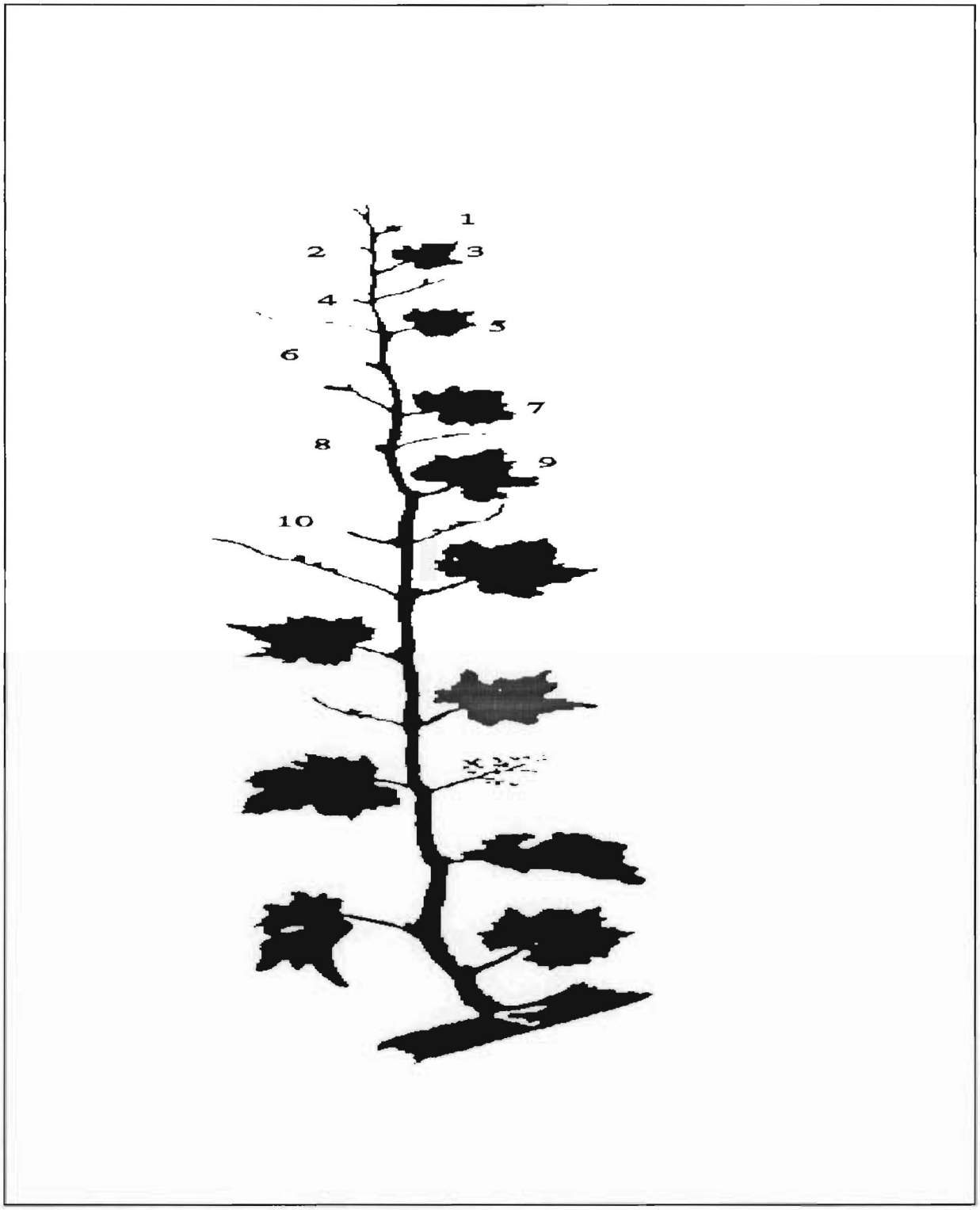


Figure 3.2
Trial 2. Alternate defoliation treatment.

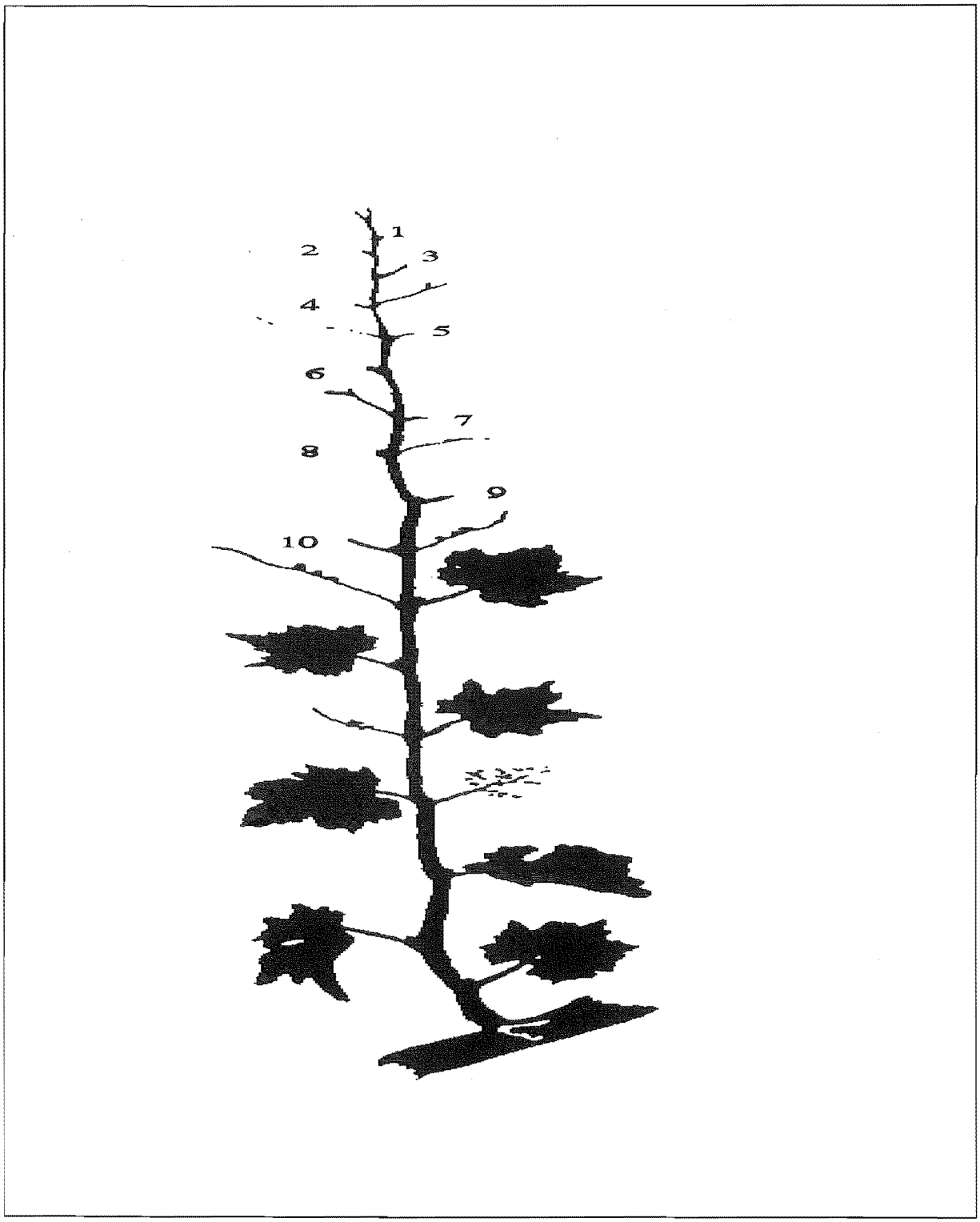


Figure 3.3
Trial 2. Complete defoliation treatment.

Plate 3.1
Mendoza Chardonnay, Lincoln University

Plate 3.2
Actively-growing Mendoza Chardonnay shoot

CHAPTER 4 RESULTS

4.1 Trial 1: C-16,17 dihydro GA_5 treatments

4.1.1 The effect of C-16,17 dihydro GA_5 on spring shoot growth

Total shoot length (ie. sum of buds 1-10) was used as a measure of the shoot growth between the C-16,17 dihydro GA_5 treatments. The number of buds lost to frost and wind damage appeared to be similar between C-16,17 dihydro GA_5 treatments.

Total shoot growth of C-16,17 dihydro GA_5 treatments were investigated at each of the weekly intervals and linear and quadratic correlations calculated (Table 4.1). The figures shown are total sum of shoot lengths for all remaining buds (ie. buds 1, 2, 4, 5, 7, 8, 9, 10) on a treated cane (ie. replicate) and then averaged between canes (replicates). The C-16,17 dihydro GA_5 treatments showed no significant effect on shoot growth in either a linear or quadratic model at any time over the five week interval.

The rate of spring shoot growth was calculated by summing the total shoot length (ie. buds 1, 2, 4, 5, 7, 8, 9, 10) for each replicate, calculating the mean shoot length per replicate and per treatment and plotting a best-fit line over time. The slope is the least-squares estimate of the line through the weekly measurement of shoot length. This is illustrated in Figure 4.1. C-16,17 dihydro GA_5 applications appear to give a greater rate of shoot growth than the control (except for 1.00 μg application which is suppressed). However, the large standard error of difference bars in Figure 4.1 show a large spread of data and indicate no significant effect of C-16,17 dihydro GA_5 on the rate of spring shoot growth.

A pair-wise comparison of intercept and slope between the nontreated and treated canes was calculated to compare the rate of shoot growth at each C-16,17 dihydro GA_5 treatment with the control. Application of C-16,17 dihydro GA_5 to the developing bud of *Vitis vinifera* in the season prior to shoot growth did not influence the rate of spring shoot growth. C-16,17 dihydro GA_5 -treated canes were not significantly different from the non-treated canes. The intercepts of the best-fit lines of shoot growth were not significantly different from the non-treated canes. Assuming spring shoot growth is linear, the applications of C-16,17 dihydro GA_5 neither promoted nor delayed bud burst.

The slopes of the lines of best fit for each treatment is shown in Table 4.2. The slope is the least-squares estimate of the line through the weekly measurement of shoot length (summed for buds 1-10). Neither the linear nor quadratic contrasts fitted the slopes at increasing concentrations of C-16,17 dihydro GA_5 treatments significantly. The quadratic model did account for the depressed rate of shoot growth in the 1.00 μg treatment but it was not significant.

Table 4.1: Trial 1 - The effects of C-16,17 dihydro GA_5 on total shoot growth (sum of buds 1-10) (mm) at weekly intervals.

TOTAL SHOOT LENGTH (MM)					
TREATMENT (μg)	**WEEK 1**	**WEEK 2**	**WEEK 3**	**WEEK 4**	**WEEK 5**
0.00	153	324	619	772	962
0.33	180	363	641	814	998
1.00	143	308	492	631	766
3.33	206	462	777	1036	1202
10.00	187	413	702	917	1108
33.33	175	348	647	812	996
SED	40.5	72.3	120.5	142.0	167.5
SIGNIFICANCE OF CONTRASTS					
Linear	n.s.	n.s.	n.s.	n.s.	n.s.
Quadratic	n.s.	n.s.	n.s.	n.s.	n.s.

Table 4.2: Trial 1 - The effects of C-16,17 dihydro GA_5 on the rate of spring shoot growth (described as the slope of the line of best-fit) for sum of buds 1-10.

TREATMENT (μg)	**SLOPE (mm/week)**
0	257
0.33	236
1.00	173
3.33	282
10.00	266
33.33	244
GRAND MEAN	243
SED	44.6
CV (%)	29.0
SIGNIFICANCE OF CONTRASTS	
Linear	n.s.
Quadratic	n.s.

4.1.2 Additional analysis of shoot growth data

Analysis of the residuals of spring shoot growth data was done to investigate any effect upon treatment results due to:

- location along the vineyard row.
- number of treatments per vine.

This was an attempt to account for some of the high variation in the data.

1. Location of treatments along the vineyard row.
   The residuals were mapped onto a vineyard row plan. No trend in the residuals was found.

2. Number of treatments per vine.
   The rate of shoot growth of each cane (ie. replicate) was divided into 'one treatment per vine' and 'multiple treatments per vine'. No trend in the mapped residuals versus fitted values was found. It appeared that treated shoots acted independently and were not affected when more than one treatment was made to a vine.

4.1.3 Log transformation of shoot growth data

The residuals were plotted against the fitted (predicted) shoot growth values. It was found that the variance was small when the shoot lengths were short but as time progressed the residual variance became greater. The data was checked for abnormal values but no specific bud or replicate was noted. The nature of the data was highly variable, with buds recording a wide range of lengths within replicates, especially by the fifth week. Log transformation of the shoot growth data did account for the increasing variance of the residuals over time, however the effect of C-16,17 dihydro GA_5 on shoot growth was no more significant. Therefore the data is presented in its natural state.

4.1.4 The effect of bud position on shoot growth

At the time of C-16,17 dihydro GA_5 application the treated buds were the last 10 which lay on the distal end of the actively growing shoot. Differentiation of the primordia occurs in the basal buds first and progresses outwards along the shoot. Thus, the 10 buds were at various stages of morphological development with bud 1 the most developed and bud 10 the most juvenile at the time of C-16,17 dihydro GA_5 application. Active growth of the vine shoots ceased soon after treatment occurred (8 February 1991). In many canes buds 9 and 10 became the terminal buds on the shoot (the term 'terminal' is used loosely as *Vitis vinifera* does not form terminal buds in the true sense). In the instances where shoot growth did continue after C-16,17 dihydro GA_5 application these buds were rubbed off the following spring to ensure that the 10 treated buds lay uniformly on the distal end of the cane and to maintain the vine at 36 buds per vine.

The occurrence of apical dominance in shoot growth along the length of the treated cane the following spring was investigated. This was to assess whether bud position (ie. buds 1-10) on the treated cane influenced total spring shoot growth in any regular trend. Jackson (1993) noted that buds at the end of a shoot may have a vigour advantage and called this the 'End Point Principle' (EPP). In many cases frost damage killed buds 9 and 10. The bud that remained beside the frosted bud was then considered terminal.

A line of best fit was calculated for total shoot growth (mm) versus bud position (ie. 1, 2, 4, 5, 7, 8, 9, 10) for each replicate of each treatment. Of the 30 replicates only 57% gave positive slopes while the remaining 43% generated negative slopes. These were evenly distributed throughout treatments with no effect of C-16,17 dihydro GA_5 concentration. Of the proportion of replicates with positive slopes only 12% showed a regular increase in total shoot length along the length of the treated cane (ie. bud 1-10). The remaining 88% although exhibiting greater total shoot length toward the distal end of the cane, contained irregularities in the shoot lengths of some buds.

Thus apical dominance was not clearly displayed by total shoot growth from the 10 distal buds on the canes. The trend in total shoot growth across the treated cane appeared random in all treatments.

4.1.5 The effect of C-16,17 dihydro GA_5 on bunch number

Bunch numbers were recorded and the effect of C-16,17 dihydro GA_5 was investigated on:

- the average number of bunches from treated buds that grew to produce a shoot (whether fruitful or not).
- the average number of bunches per fruitful shoot.
- the incidence of fruitless shoots throughout treatments (described as %) from treated buds.

C-16,17 dihydro GA_5 had no effect on bud fertility the season following application (Table 4.3)

Table 4.3: Trial 1 - The effects of C-16,17 dihydro GA_5 on the number of bunches produced the following season

TREATMENT (μg)	AVERAGE BUNCH NUMBER PER GROWING SHOOT	AVERAGE BUNCH NUMBER PER FRUITING SHOOT	% FRUITLESS SHOOTS
0.00	1.26	1.62	20
0.33	1.34	1.71	21
1.00	1.18	1.83	37
3.33	1.32	1.65	19
10.00	1.43	1.67	15
33.33	1.35	1.64	17
GRAND MEAN	1.31	1.69	21
SED	0.266	0.234	12.4
CV %	30.8	21.9	9.2
SIGNIFICANCE OF CONTRASTS			
Linear	n.s.	n.s.	n.s.
Quadratic	n.s.	n.s.	n.s.

4.1.6 Interaction between bud location and defoliation on bunch number

Inflorescence numbers of buds 1 and 2 were summed for each treatment as were buds 9 and 10. Buds 1 and 2 were older and more developed than buds 9 and 10 at the time of application (Refer to Figure 3.1). In the instance where the treated bud was missing (frost or wind damage) the inflorescence number was taken from the next treated bud along that produced a shoot.

There was no significant effect of C-16,17 dihydro GA_5 treatments on the bunch numbers and no interaction between C-16,17 dihydro GA_5 and location of the buds on the cane (ie 'older' proximal buds versus 'younger' distal buds) in the number of bunches formed (Refer Section 3.5.5.). There was however, a significant difference in the number of bunches formed due to location on the cane (significant to the 5% level). Proximal buds, which were older, gave a higher mean number of inflorescence than distal buds which developed later in the season.

When treatments were analysed individually for the effect of bud location (and hence bud age) on bunch number each C-16,17 dihydro GA_5 treatment gave greater bunch numbers in the proximal buds compared to the distal buds (Table 4.4). Only at the 3.33 μg C-16,17 dihydro GA_5 treatment was the mean distal number of bunches significantly different than the proximal (to 4.9%). In virtually all cases the proximal bunch numbers were greater than the distal buds.

Table 4.4: Trial 1 - The effects of bud location on the number of bunches formed on C-16,17 dihydro GA_5 treated canes.

TREATMENT (μg)	PROXIMAL BUNCH NUMBER (BUDS 1+2)	DISTAL BUNCH NUMBER (BUDS 9+10)
0.00	2.20	2.20
0.33	3.20	2.00
1.00	1.80	1.60
3.33	3.20	1.40
10.00	3.20	2.80
33.33	3.20	2.00
GRAND MEAN	2.80	2.00
SED compared treatments		0.95
SED compared grand means		0.38

4.1.7 The effect of C-16,17 dihydro GA_5 on bunch mass

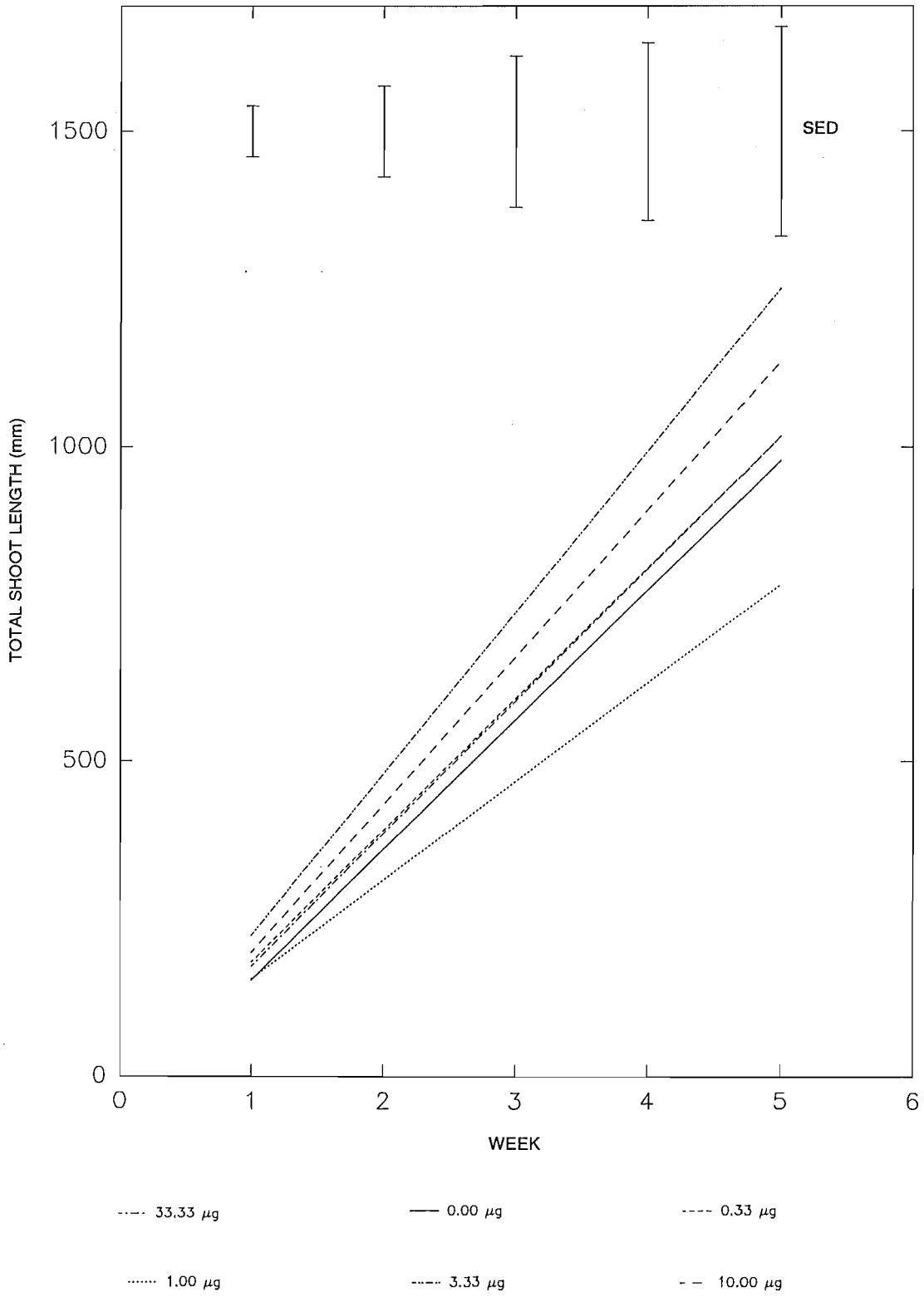
Bunch mass was measured as grams per shoot (ie. the mass of fruit produced by a treated bud that successfully grew the spring following C-16,17 dihydro GA_5 application). Buds that produced a shoot but no fruit were recorded as having a fruit mass of 0.0g as the potential for fruit remained. Average fruit mass (g) per growing shoot was favoured to total fruit mass per treated cane (ie. sum of buds 1-10) as missing buds (lost to frost and wind damage) reduced the potential harvest of some canes. The mean mass per growing shoot accounts for the mass of fruit harvested from buds that actually grew. The results are presented in Table 4.5.

C-16,17 dihydro GA_5 had no effect on the mass of fruit produced per treated bud that successfully grew the following spring.

Table 4.5: Trial 1 - The effect of C-16,17 dihydro GA_5 on fruit mass

TREATMENT (μg)	MEAN FRUIT MASS (g) PER FRUITFUL SHOOT
0.00	26.35
0.33	22.76
1.00	30.42
3.33	30.24
10.00	30.28
33.33	24.76
GRAND MEAN	27.47
SED	10.15
CV %	58.4

Figure 4.1. The effects of C-16, 17 dihydro GA5 on the rate of spring shoot growth.



4.2 Trial 2: Defoliation and ethephon Treatments

4.2.1 The effect of defoliation on shoot growth

The slopes of spring shoot growth were calculated as in Section 4.1.1 and are presented in Table 4.6 and Figure 4.2. The slopes are the sum total of shoot growth (mm) for buds 1-10 at each weekly interval. Both the partial and total defoliation treatments gave a greater, though non-significant rate of spring shoot growth (slopes) than the non-treated canes.

The mean shoot lengths at week five were calculated as in Section 3.5.5. for proximal and distal buds. When only proximal and distal buds were analysed defoliation had no effect on shoot length. At each defoliation treatment (ie. control, alternate and total defoliation) mean shoot lengths of proximal buds were greater than distal buds, although this was not significant ($p=0.14$). No interaction of bud location and defoliation on shoot length was found.

4.2.2 The effect of defoliation on bunch numbers

Although not significant, defoliation reduced the mean bunch number per fruiting shoot (Table 4.6). The alternately defoliated vines gave a mean bunch number per fruiting shoot mid way between the non-treated and the total defoliation vines.

In each treatment the sum of bunches in the proximal buds were greater than the distal buds (Refer Section 3.5.5). Over all treatments the proximal buds gave significantly greater bunch numbers than the distal buds ($p=<0.001$). No effect of defoliation on bunch numbers was found when only the proximal and distal buds were utilised on each cane. There was no interaction between defoliation and bud location on bunch numbers.

4.2.3 The effect of defoliation on bunch mass

Although not significantly different, the mean fruit mass per shoot decreased with increasing defoliation (Table 4.6).

Over all three treatments the mean fruit mass (g) of the proximal buds was significantly greater than the distal buds ($p = < 0.10$). No effect of defoliation was found within only proximal and distal buds, although fruit mass declined with increasing defoliation within the proximal buds. There was no interaction between bud location (Refer Section 3.5.5) and defoliation on fruit mass.

Table 4.6: Trial 2: The effects on vegetative and fruiting characteristics the season following defoliation.

TREATMENT	SLOPE (mm/Week) (sum of buds 1-10)	MEAN BUNCH NUMBER PER FRUITING SHOOT	MEAN FRUIT MASS (G) PER FRUITING SHOOT
CONTROL	239	1.78	42.86
ALTERNATE DEFOLIATION	322	1.68	41.62
TOTAL DEFOLIATION	317	1.58	31.68
GRAND MEAN	291	1.68	38.72
SED	56.56	0.167	14.75
CV %	32.7	16.25	62.21
SIGNIFICANCE OF CONTRASTS			
Control vs (Alternate+Total)/2	p=0.105	n.s.	n.s.
Alternate vs Total	n.s.	n.s.	n.s.

4.2.4 The effect within the alternately defoliated treatment on shoot growth

An analysis of variance of the Week 5 shoot lengths found no interaction of bud location in the alternately defoliated treatment (Refer Section 3.5.5). Shoot lengths were then meaned over the canes and presented in Table 4.7. At each week the mean shoot lengths of the 'removed leaf' buds were greater than the 'intact leaf'. The increase in shoot growth with removal of the subtending leaf repeated the trend found between defoliation treatments in Section 4.2.1.

The slope of shoot growth was calculated for the mean shoot lengths of 'intact leaf' (buds 1, 3, 5, 7, 9) and 'removed leaf' (buds 2, 4, 6, 8, 10) treatments over the five week interval. The analysis of variance of these slopes is presented in Table 4.8.

When all six replicates were included no significant difference between the slopes of the two treatments was found, although the mean slope of the 'removed leaf' treatment was greater than the 'intact leaf'. When the sixth replicate was removed from the data the slope of the 'removed leaf' treatment was significantly greater than the 'intact leaf' treatment ($p = < 0.10$). The 'removed leaf' buds in the sixth replicate exhibited a greater proportion of latent buds, thus disproportionately lowering the rate of shoot growth.

With a greater replicate number possibly this trend (localised to one peculiar cane) would not have had such an influence as it would have accounted for a smaller proportion of the total data set. In contrast, with a greater replicate number more trends in shoot growth typical of replicate six may have occurred and no difference between intact and defoliated buds found.

When the sixth replicate is removed it enhances results found in the mean values when all replicates are included. It also repeats the trend of increased shoot growth found in defoliated treatments in Section 4.2.1.

Table 4.7: Trial 2: Mean shoot length per bud (mm) over a five week interval of the alternately defoliated treatment.

	BUD POSITION (MEAN SHOOT LENGTH PER BUD MM)												
	1 I	2 R	3 I	4 R	5 I	6 R	7 I	8 R	9 I	10 R	WEIGHTED MEAN INTACT	WEIGHTED MEAN REMOVED	SED
WK 1	31.7	38.3	43.3	52.5	38.0	57.0	38.3	50.8	34.0	21.0	37.9	44.5	8.17
WK 2	60.0	69.2	81.7	100.0	74.0	124.0	78.3	96.7	74.0	46.0	75.1	86.2	15.08
WK 3	101.7	112.5	143.3	148.0	108.0	190.0	118.0	145.0	132.0	70.0	126.8	131.5	26.75
WK 4	135.0	132.5	170.0	188.0	134.0	226.0	132.0	173.3	148.0	78.0	150.8	157.4	30.58
WK 5	158.3	165.8	210.0	230.0	164.0	274.0	170.0	206.7	168.0	96.0	183.8	191.5	37.48

'I' = Intact
'R' = Removed

4.2.5 The effect within the alternately defoliated treatment on bunch number

Defoliated buds produced less bunches the following season than nontreated buds ($p = < 0.10$). Data are presented in Table 4.8 and repeat the trend found in Section 4.2.2.

4.2.6 The effect within the alternately defoliated treatment on bunch mass

Within the alternate defoliation treatment the mean fruit mass (g) of buds with the subtending leaf intact was significantly greater than defoliated buds ($p = < 0.10$). Data are presented in Table 4.8. This repeats the trend found in Section 4.2.3 between the non-treated, alternate and total defoliation fruit mass means.

Table 4.8: Trial 2: Effects of removing alternate subtending leaves within a cane on the vegetative and fruiting characteristics the following season.

TREATMENT	SLOPE (mm/week/bud)		MEAN BUNCH NUMBER PER SHOOT	MEAN FRUIT MASS PER SHOOT (g)
	SIX REPLICATES	FIVE REPLICATES		
INTACT LEAF BUD	36.7	32.3	1.77	51.71
REMOVED LEAF BUD	37.9	38.4	1.32	33.55
GRAND MEAN			1.54	43.04
SED	9.40	4.06	0.356	9.45
CV (%)	25.2	11.5	23.03	73.18
SIGNIFICANCE OF DIFFERENCE	n.s.	$p = < 0.10$	$p = < 0.10$	$p = < 0.10$

4.2.7 Interaction of bud location and defoliation on bunch mass

Mean fruit mass (g) of the proximal buds was significantly greater than the distal buds ($p=0.01$). There was no interaction between the effects of defoliation and location on the cane on fruit mass.

Table 4.9 presents the effect of bud location and defoliation on fruit mass within the alternately defoliated treatment. The mean fruit mass was depressed in the distal buds and further depressed when the subtending leaf was removed.

Table 4.9: The effects of bud location and defoliation on mean fruit mass (g) per growing shoot.

TREATMENT	MEAN FRUIT MASS PER SHOOT (g)
PROXIMAL INTACT	63.33
PROXIMAL REMOVED	42.83
DISTAL INTACT	27.93
DISTAL REMOVED	11.58
GRAND MEAN	36.42
SED	11.19

4.2.8 The effect of ethephon

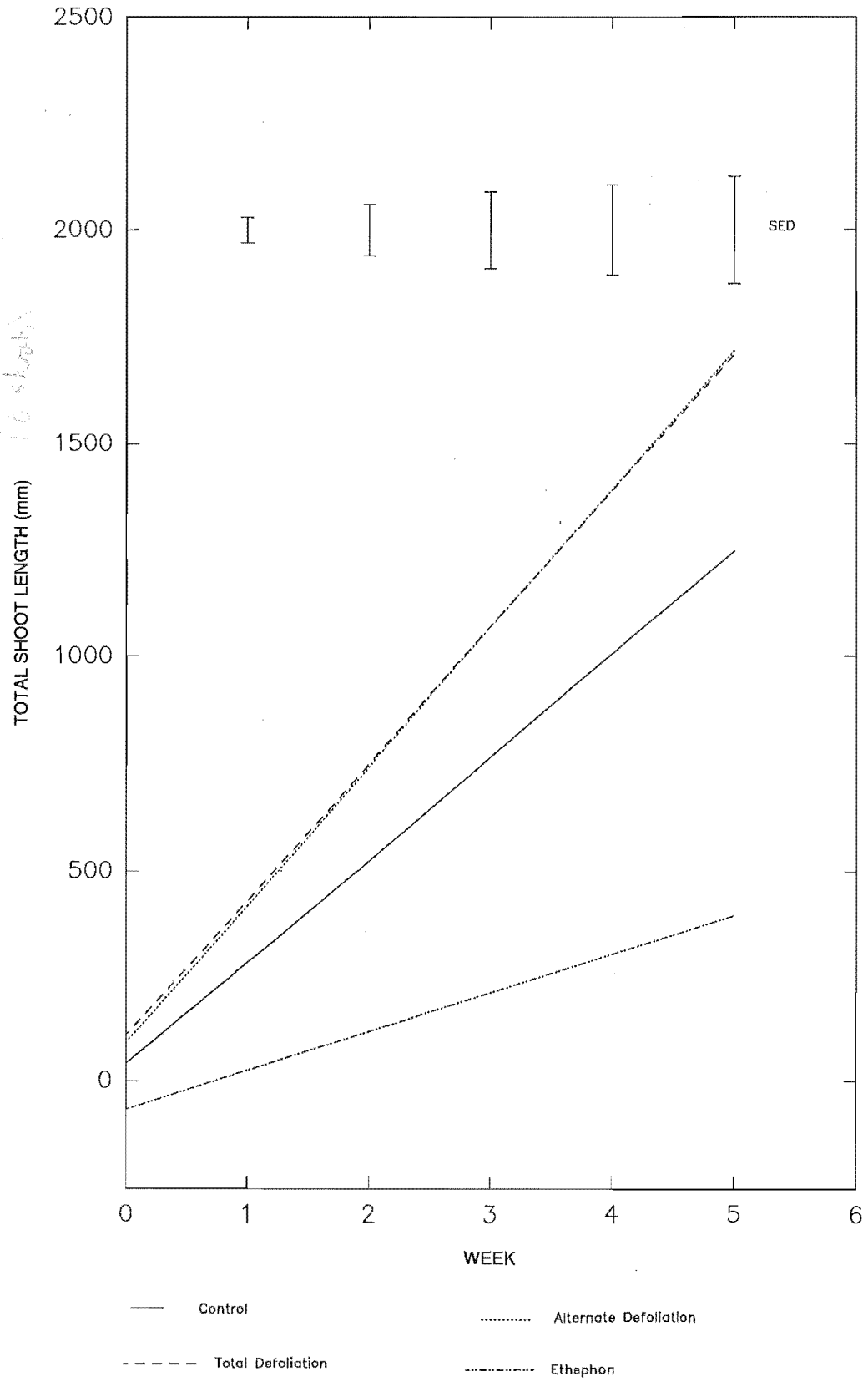
A line of best fit for the rate of shoot growth over the five week interval was calculated for each treatment in Trial 2 and presented in Figure 4.2. The application of ethephon to actively-growing shoots reduced the rate of shoot growth the following spring. At week five the mean shoot length of the remaining buds in the ethephon treatment was less than the non-treated buds (Table 4.10).

Table 4.10: The effects of ethephon (500ppm) on shoot growth the following spring.

TREATMENT	MEAN SHOOT LENGTH (mm)
Non-treated (n=39)	186
Ethephon (n=6)	102

It was noted in visual inspections that all active growth of ethephon-treated canes had ceased within a week of application. Epinasty (downward curvature) of the shoots and leaves was followed by death of foliage and shoot tips. The following spring only three of the six replicates recorded any shoot growth from treated buds. In the remaining three replicates shoots grew only from the first three, first two and second bud/s respectively. The buds that did produce shoots grew more slowly than the non-treated canes. Further analyses of the effect of ethephon on bunch numbers and fruit mass was not performed due to an excessive amount of missing data.

Figure 4.2. The effects of defoliation and ethephon treatment on the rate of spring shoot growth.



CHAPTER 5 DISCUSSION

5.1 The effects of C-16,17 dihydro GA_5 applications

The application of C-16,17 dihydro GA_5 had no effect on shoot growth, bunch number or bunch mass the spring following application to developing axillary buds (refer to Tables 4.2, 4.3, 4.5). Previous experimentation with C-16,17 dihydro GA_5 retarded shoot growth and induced inflorescence initiation in some annual crops (Pharis *et al.*, 1993).

Crozier (1983) noted various successful methods of application of gibberellins, including aqueous solutions of GA applied to shoot tips and/or young leaves. As C-16,17 dihydro GA_5 is a gibberellic acid derivative, application in similar fashion to other Gas seems sensible. Thus, it appears unlikely the method of application by microdrops of aqueous solution directly onto developing axillary buds is responsible for any lack of response in this trial.

The chance of incorrect concentrations of C-16,17 dihydro GA_5 was reduced by application in logarithmic concentrations. Thus, any effect of C-16,17 dihydro GA_5 at a specific level could be interpolated. However, in a perennial vine the concentrations of C-16,17 dihydro GA_5 required for a significant effect may possibly be greater.

With the existence of many gibberellins, Crozier (1983) noted it to be conceivable for a certain degree of species-specificity in regard to flower-inducing capacity. As only a few gibberellins occur in each species, failure to induce flowering may be due to the application of the "wrong" gibberellin. This could explain the nil effect of C-16,17 dihydro GA_5 on *Vitis vinifera* in this trial.

C-16,17 dihydro GA_5 at 1.00 μg gave some unusual results, although non were statistically significant. Suppression of the rate of spring shoot growth (Table 4.2), a high bunch number per fruiting shoot (Table 4.3) and a high incidence of % fruitless shoots (Table 4.3). The author notes this is most probably due to unfortunate random selection of less-developed shoots. Thomas and Barnard (1937) found immaturity in the shoot to be associated with a low starch content and consequent poor development of anlagen.

Overall, the high variance in the data and missing buds (due to frost and wind damage) weakened the integrity of the experiment. A greater number of replicates and application of C-16,17 dihydro GA_5 earlier in the season (to minimise frost damage to treated buds) may have given the experiment greater robustness. However, extensive analysis concluded C-16,17 dihydro GA_5 had no effect on *Vitis vinifera*, cv. Chardonnay in this trial.

5.2 The effect of defoliation on inflorescence initiation

Defoliation of developing buds suppressed inflorescence initiation relative to the non-defoliated canes although this was not statistically significant. The alternately-defoliated vines gave a mean bunch number per fruiting shoot midway between the non-treated and totally defoliated treatments (Table 4.6). Within the alternately-defoliated treatment the suppression of inflorescences was repeated. Fewer bunches were produced on the shoot the season following defoliation of the developing axillary bud. These results were significant at the 10% level (Table 4.8). Minnis (1970) working with apricots, also found removal of the leaf subtending a developing bud to inhibit floral initiation. He stated the earlier in the season the subtending leaf was removed the greater was the effect of reduced fertility.

The suppression of inflorescence initiation between and within canes suggests a real effect of defoliation, localised at the bud specifically treated. Lavee *et al.* (1976) postulated that the stimulus in grape vines for flower initiation came from the leaves located at and above the examined bud. They found no evidence of the vine "pooling" the stimulus produced in the leaves for the rest of the vine.

5.3 The effect of defoliation on bunch mass

Defoliation of developing buds suppressed the mass of fruit produced the following season, although this was not statistically significant (Table 4.6). Within the alternately-defoliated treatment fruit mass of defoliated buds was also reduced, significant at the 10% level (Table 4.8). Barnard and Thomas (1938) found in years of low bud fertility the average bunch weight tends to be greater than when a larger number of bunches are to be matured. This is due primarily to an increase in berry size. In this trial any possible increase in berry size was not great enough to nullify the effect of reduced bud fertility with defoliation. Barnard and Thomas (1932) noted the potential size of inflorescences to be determined by the amount of growth made by the primordia prior to bud burst in spring. The author suggests the reduction in fruit mass of defoliated buds is a result of a lack of development of the inflorescence (cluster) primordia at the onset of dormancy due to the loss of the main photosynthate and/or hormonal source, the leaf subtending the bud.

5.4 The effect of defoliation on shoot growth

Defoliation (both alternate and total) of developing buds increased the rate of shoot growth the following spring, although this was not statistically significant (Table 4.6 and Figure 4.2). Within the alternately-defoliated treatment, buds with subtending leaf removed showed an increase in the rate of shoot growth ($p = < 0.10$) and greater total shoot length over the first five weeks (Tables 4.7, 4.8).

The acceleration of shoot growth with defoliation is possibly the result of vegetative shoots growing at a faster rate than fruitful shoots. Less energy is spent on developing clusters, and as a consequence the rate of shoot growth is greater. Brundell (1975) working with kiwifruit found defoliating the axillary bud to promote shoot growth and debated whether hormonal factors present in leaves and/or an enhanced attraction of metabolites to the shoot apex were responsible.

5.5 The influence of bud location on the effect of defoliation

In this trial no interaction of bud location (proportional to bud age) and defoliation was found. It may be postulated however, that the younger the bud the greater the effect of defoliation on bud fertility.

The reduction in fruitfulness and fruit mass in the distal buds across all defoliation treatments may represent the terminal section of a quadratic trend in fruitfulness (Lavee *et al.*, 1976;, May, 1973) and cluster mass (Winkler and Shemsettin, 1937). Thomas and Barnard (1937) found the quadratic trend in bud fruitfulness to be repeated in a quadratic trend of cane starch content. They suggested reduction in carbohydrate storage in the distal portion of the cane would account for reduction in bud fertility and yield in the subsequent season. Buttrose (1969b, 1970) investigating the effect of temperature on bud fertility found buds responded directly to high maximal temperatures during an early stage of development. They also found the "hours of sunshine" in the season prior were also closely related to yield the following season. As a result of treatment application later in the summer (8 February) the younger (distal) buds developed in an environment of increasingly shorter day lengths and cooler temperatures. This may have reduced bud fertility directly, or, encompassing Thomas and Barnard (1937) theory, was a result of lower starch storage in the cane.

Hale and Weaver (1962) noted that leaves became exporters of assimilates when they were about half of their mature size. Thus the younger leaves in the defoliation trials were initially metabolic sinks, probably competing directly with the developing axillary bud. Defoliated young buds would have required carbohydrates for all their differentiation. This would (assumingly) have been supplied by the leaves below. When these older (proximal) leaves were also defoliated the source/s of carbohydrates were reduced and competition from other developing axillary buds increased. It might have been expected that the younger (distal) buds would be less fruitful with defoliation than the older buds, which had the advantage of exploiting their subtending leaves during the critical stage of inflorescence initiation.

The lack of any interaction in this trial did not confirm this idea and may have been a consequence of treatment application too late in the growing season. It is also possible assimilates from leaves further down the cane were translocated up to the treated buds. However, the range of 10-treated leaves was felt to encompass a fair representation of both net importing and exporting leaves. Repetition of the experiment earlier in the summer and with a greater number of replicates may strengthen the trial and indicate any trend in fertility with defoliation relative to bud age. Measuring the size of the leaf at the time of defoliation may also allow predictions on its assimilate status (ie. source or sink).

5.6 Mechanisms determining inflorescence initiation

Two theories are offered in the literature to account for the mechanism of inflorescence initiation in *Vitis vinifera*. These include direction by the presence of hormones and control by carbohydrate supply.

Palma (1985) considered inhibition or promotion of inflorescence in grape vines as the result of hormones and not solely carbohydrate supply. He suggested that the relatively minute inflorescence anlage would require very small amounts of carbohydrates (relative to the entire vine) to successfully initiate inflorescence. Thus the mechanism was felt to be under hormonal direction. Srinivasan and Mullins (1980c) had previously suggested this, proposing climatic effects were mediated through consequent alterations in endogenous plant hormones to directly influence inflorescence initiation.

However popular the concept of floral initiation by hormone stimuli, identification of a specific compound has not been achieved. Support for the concept (as suggested by Crozier, 1983) of a sole compound (florigen) has declined in favour of possible interactions by several endogenous hormones (Jackson and Sweet, 1972;, Zeevart, 1976). Identification of floral-inducing hormones is tentative although literature suggests that translocation appears to be limited (Lavee *et al.*, 1976;, Jackson and Sweet, 1972;, Buttrose, 1969b;, Minnis, 1970). Minnis (1970) surmised that inflorescence initiation was directed by hormones produced in small quantities locally in the leaf.

The development of inflorescence primordia is a growth process positively correlated to vine vigour (except in extremely vigorous sites) (Thomas and Barnard, 1937). Carbohydrate accumulation is recognised as a condition necessary for successful differentiation and is dependent upon growth rate prior to winter dormancy and sufficient maturation of canes (Lavee *et al.*, 1976;, Botti and Sandoval, 1990;, May, 1965;, Srinivasan and Mullins, 1980a;, Thomas and Barnard, 1937). Although axillary buds are considered a relatively weak sink for carbohydrates, the chief source of photosynthates appears to be the leaf subtending it (Brundell, 1975;, Hale and Weaver, 1962).

This trial suggests the factor responsible for inflorescence initiation may be localised in the leaf subtending the axillary bud. Minnis (1970) similarly concluded the factor in the leaf was either generated in small amounts throughout the time of initiation or else not very mobile in the plant. It is the author's opinion that the results of this trial suggest the presence of an endogenous hormone localised in the leaf subtending the developing axillary bud. A rationale of inflorescence initiation suggested by Sachs (1977) and favoured by the author incorporates both hormonal and carbohydrate strategies. He viewed inflorescence initiation as a result of the modification of metabolic supplies and distribution to differentiating anlagen by chemical (hormonal) changes influenced by the environment.

5.7 The effects of ethephon

Ethephon application (as 'Ethrel' at 500 ppm a.i. ethephon) on February 8 killed most of the axillary buds that were treated. The few buds that survived and grew the following spring did so at a lower rate than the non-treated buds (Table 4.10). Although the rate applied in this trial is reasonably low for a commercial application, it would appear to be too high to study the effects on developing buds. To compound this, possible over-application may have occurred with the hand held sprayer. Microdrop applications of ethephon in various strengths specifically onto differentiating buds may allow more successful future investigations.

5.8 Conclusion

In this investigation both trials involved treating the first 10 buds of actively-growing *Vitis vinifera* shoots. Axillary buds develop in acropetal order along the length of the cane. Thus, the first 10 buds were at various stages of differentiation when treatment occurred.

The application of C-16,17 dihydro GA_5 by microdrops directly onto developing axillary buds had no effect the subsequent season. Pharis *et al.* (1993) suggested failure to observe organ elongation and/or promotion of flowering by C-16,17 dihydro GAs was due to wrong choices of assay systems. It would appear, assuming the assay system is appropriate, that C-16,17 dihydro GA_5 has no effect on the development of differentiating buds in *Vitis vinifera*. The author suggests Crozier's (1983) concept of *species specificity* may explain the nil effect in this trial.

In the second trial the reduced inflorescence initiation and fruit mass and promotion of shoot growth suggests a real effect of defoliation. Results within the alternately defoliated treatment suggest the mechanism responsible is localised at the leaf subtending the developing bud. Although the results were seldom statistically significant (and those that were, no more than the 10% level) trends were consistently repeated both between defoliation treatments and within the alternately-defoliated treatment. This suggests a real result, overriding any effect wind and frost damage had to the vines. Previous experimentation with grapevines and apricots has noted the importance of leaves located at and above the examined bud, with no evidence of the vine "pooling" the stimulus throughout the plant (Lavee *et al.*, 1976; Minnis, 1970). This may suggest endogenous plant hormones produced in the leaf subtending the developing bud governs inflorescence initiation in *Vitis vinifera*.

Ethephon application to actively-growing shoots of *Vitis vinifera* unfortunately destroyed most of the developing axillary buds. This made measurements of fruitfulness and vegetative growth infeasible. The author suggests in the future that lower concentrations of ethephon be applied, perhaps directly onto the axillary bud.

FURTHER RESEARCH

The results from the C-16,17 dihydro GA_5 trial indicates there is no effect on fruitfulness or vegetative development the season following application to *Vitis vinifera*. The author suggests that if future trials were to be undertaken applications should be made earlier in the summer (to avoid frost damage) and with a greater number of replicates, to reduce variability in the data.

The reduction in bud fruitfulness with defoliation helps to define the role of the subtending leaf during differentiation of the axillary bud. Future research may include:

- Estimating the *age* of the leaf at which time defoliation has the greatest impact in the fertility of the bud. This could lead to predictions of *safe* leaf-plucking heights in the canopy and ensure maximum light penetration while maintaining bud fertility.

- Possible identification of endogenous hormone/s that the subtending leaf possesses/generates.

- The effect that loss of developing-leaf area from wind damage, water stress, close trimming and pest/pathogen damage has on bud fertility the following season.

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